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APPLICATION NO.	FILING DATE	FIRST NAMED INVENTOR	ATTORNEY DOCKET NO.	CONFIRMATION NO.
10/583,860	05/21/2007	Takashi Nishimura	3691-0133PUS1	8593

2292 7590 05/13/2010

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EXAMINER	
CHEN, SHIN LIN	
ART UNIT	**PAPER NUMBER**
1632	

NOTIFICATION DATE	DELIVERY MODE
05/13/2010	ELECTRONIC

Please find below and/or attached an Office communication concerning this application or proceeding.

The time period for reply, if any, is set in the attached communication.

Notice of the Office communication was sent electronically on above-indicated "Notification Date" to the following e-mail address(es):

mailroom@bskb.com

Office Action Summary	Application No.	Applicant(s)	
	10/583,860	NISHIMURA ET AL.	
	Examiner	Art Unit	
	Shin-Lin Chen	1632	

-- The MAILING DATE of this communication appears on the cover sheet with the correspondence address --

Period for Reply

A SHORTENED STATUTORY PERIOD FOR REPLY IS SET TO EXPIRE *3* MONTH(S) OR THIRTY (30) DAYS, WHICHEVER IS LONGER, FROM THE MAILING DATE OF THIS COMMUNICATION.

- Extensions of time may be available under the provisions of 37 CFR 1.136(a). In no event, however, may a reply be timely filed after SIX (6) MONTHS from the mailing date of this communication.
- If NO period for reply is specified above, the maximum statutory period will apply and will expire SIX (6) MONTHS from the mailing date of this communication.
- Failure to reply within the set or extended period for reply will, by statute, cause the application to become ABANDONED (35 U.S.C. § 133). Any reply received by the Office later than three months after the mailing date of this communication, even if timely filed, may reduce any earned patent term adjustment. See 37 CFR 1.704(b).

Status

1)☒ Responsive to communication(s) filed on *09 March 2010*.

2a)☐ This action is **FINAL**. 2b)☒ This action is non-final.

3)☐ Since this application is in condition for allowance except for formal matters, prosecution as to the merits is closed in accordance with the practice under *Ex parte Quayle*, 1935 C.D. 11, 453 O.G. 213.

Disposition of Claims

4)☒ Claim(s) *1,3-9 and 11-22* is/are pending in the application.

4a) Of the above claim(s) *6,14 and 18-21* is/are withdrawn from consideration.

5)☐ Claim(s) _____ is/are allowed.

6)☒ Claim(s) *1,3-5,7-9,11-13,15-17 and 22* is/are rejected.

7)☐ Claim(s) _____ is/are objected to.

8)☐ Claim(s) _____ are subject to restriction and/or election requirement.

Application Papers

9)☐ The specification is objected to by the Examiner.

10)☐ The drawing(s) filed on _____ is/are: a)☐ accepted or b)☐ objected to by the Examiner.

Applicant may not request that any objection to the drawing(s) be held in abeyance. See 37 CFR 1.85(a).

Replacement drawing sheet(s) including the correction is required if the drawing(s) is objected to. See 37 CFR 1.121(d).

11)☐ The oath or declaration is objected to by the Examiner. Note the attached Office Action or form PTO-152.

Priority under 35 U.S.C. § 119

12)☐ Acknowledgment is made of a claim for foreign priority under 35 U.S.C. § 119(a)-(d) or (f).

a)☐ All b)☐ Some * c)☐ None of:

1.☐ Certified copies of the priority documents have been received.

2.☐ Certified copies of the priority documents have been received in Application No. _____.

3.☐ Copies of the certified copies of the priority documents have been received in this National Stage application from the International Bureau (PCT Rule 17.2(a)).

* See the attached detailed Office action for a list of the certified copies not received.

Attachment(s)

1) ☒ Notice of References Cited (PTO-892)
2) ☐ Notice of Draftsperson's Patent Drawing Review (PTO-948)
3) ☐ Information Disclosure Statement(s) (PTO/SB/08) Paper No(s)/Mail Date _____.
4) ☐ Interview Summary (PTO-413) Paper No(s)/Mail Date. _____ .
5) ☐ Notice of Informal Patent Application
6) ☐ Other: _____.

DETAILED ACTION

1. A request for continued examination under 37 CFR 1.114, including the fee set forth in 37 CFR 1.17(e), was filed in this application after final rejection. Since this application is eligible for continued examination under 37 CFR 1.114, and the fee set forth in 37 CFR 1.17(e) has been timely paid, the finality of the previous Office action has been withdrawn pursuant to 37 CFR 1.114. Applicant's submission filed on 3-9-10 has been entered.

Applicant's amendment filed 3-9-10 has been entered. Claims 1, 3, 4, 9, 11, 12 and 18-21 have been amended. Claim 22 has been added. Claims 1, 3-9 and 11-22 are pending. Claims 1, 3-5, 7-9, 11-13, 15-17 and 22 and the species WT1 are under consideration.

Claim Objections

2. Claim 7 is objected to because of the following informalities: The phrase "for cell therapy according to any of claims 1, 3, 4 or 6" in claim 7 recites claim 6, which is a non-elected claim. Deleting "or 6" in claim 7 would be remedial. Appropriate correction is required.

3. Claim 15 is objected to because of the following informalities: The phrase "for cell therapy according to any of claims 9, 11, 12 or 14" in claim 15 recites claim 14, which is a non-elected claim. Deleting "or 14" in claim 15 would be remedial. Appropriate correction is required.

Claim Rejections - 35 USC § 102

4. The following is a quotation of the appropriate paragraphs of 35 U.S.C. 102 that form the basis for the rejections under this section made in this Office action:

> A person shall be entitled to a patent unless –
>
> (b) the invention was patented or described in a printed publication in this or a foreign country or in public use or on sale in this country, more than one year prior to the date of application for patent in the United States.

5. Claims 1, 4 and 7 are rejected under 35 U.S.C. 102(b) as being anticipated by Fujio et al., 2000 (Journal of Immunology, Vol. 165, p. 528-532).

Claims 1, 4 and 7 are directed to a process of preparing cells for cell therapy comprising inducing helper T cells that have a nonspecific antitumor activity, and imparting antigen specificity to the helper T cells by transducing the helper T cells with a T cell receptor gene that recognizes a cancer-associated antigen, wherein the TCR gene is a MHC class II-restricted T cell receptor gene. Claim 7 specifies further purifying the Th1 cells to which antigen specificity has been imparted.

Fujio teaches that "transfer of the alphabeta TCR genes into T lymphocytes will provide a means to enhance Ag-specific immunity by increasing the frequency of tumor- or pathogen-specific T lymphocytes. Fujio co-transfect TG40 cells, a TCR-negative mouse T cell line, with retroviral vector expressing either of the class II MHC-restricted alpha or beta TCR gene specific for chicken OVA and results in expression of the clonotypic TCR in 44% of the CD4+ T cells (helper T cells). "The transduced cells showed a remarkable response to OVA323-339 peptide in the in vitro culture system ... Adoptive transfer of the TCR-transduced cells in mice induced the Ag-specific delayed-type hypersensitivity in response to OVA323-339 challenge" (e.g. abstract, p. 528, right column, last paragraph). The TG40 cells are considered induced helper T cells. The chicken OVA is considered a cancer-associated antigen. Thus, the claims are anticipated by Fujio.

6. Claims 1, 3, 7, 9, 11 and 15 are rejected under 35 U.S.C. 102(b) as being anticipated by Kessels et al., 2001 (Nature Immunology, Vol. 2, No. 10, p. 957-961).

Claims 1, 3 and 7 are directed to a process of preparing cells for cell therapy comprising inducing helper T cells that have a nonspecific antitumor activity, and imparting antigen specificity to the helper T cells by transducing the helper T cells with a T cell receptor gene that recognizes a cancer-associated antigen, wherein the TCR gene is a MHC class I-restricted T cell receptor gene. Claim 7 specifies further purifying the Th1 cells to which antigen specificity has been imparted. Claims 9, 11 and 15 are directed to a process of preparing cells for cell therapy comprising inducing Th1 cells and Tc1 cells having a nonspecific antitumor activity and imparting antigen specifically to the Th1 cells and Tc1 cells by transducing the Th1 cells and Tc1 cells with a TCR gene that recognizes a cancer-associated antigen. Claim 11 specifies transducing with MHC class I-restricted TCR gene. Claim 15 further comprises a step of separating the Th1 cells and Tc1 cells to which antigen specificity has been imparted.

Kessels teaches that "the antigen specificity of T lymphocytes is dictated solely by the T cell receptor (TCR) alpha and beta chains. Consequently, genetic transfer of TCR chains may be an appealing strategy with which to impose a desirable virus- or tumor-antigen specificity onto cytotoxic or helper T cell populations" (e.g. abstract). Kessels introduces F5 TCR chains into mouse spenocytes by retroviral infection and 5-15% of the total CD8+ T cells (cytotoxic T cells) expressed the F5 TCR. The F5 TCR-transduced T cells show a pronounced NP(366-374)-specific effector function and the retroviral introduction of TCRs leads to rapid generation of antigen-specific T cell immunity (e.g. p. 958, left column, 2nd paragraph). Kessels also introduces F5 TCR chains into total spleen cells, including CD4+ T cells with pMX-F5 virus and

5-10% of the CD4+ T cells expressed the F5 receptor. However, "in contrast to the marked expansion of F5 TCR CD8+ T cells upon influenza A/NT/60/68 infection, no expansion of F5 TCR CD4+ T cells was detected…This lack of expansion of TCR-transduced CD4+ T cells was consistent with the idea that CD4 coreceptor binding and signaling is required for proper T cell activation; it suggests that, for the simultaneous induction of CD4+ T cell immunity, coapplication of retrovirus encoding MHC class II-restricted TCRs may be considered" (e.g. p. 959, left column, 2nd paragraph). Imparting antigen specificity to the Th1 cells and Tc1 cells only requires transducing the Th1 cells and Tc1 cells with a TCR gene that recognizes a cancer-associated antigen. F5 TCR is a MHC class I-restricted TCR. Thus, the claims are anticipated by Kessels.

Claim Rejections - 35 USC § 103

7. The following is a quotation of 35 U.S.C. 103(a) which forms the basis for all obviousness rejections set forth in this Office action:

> (a) A patent may not be obtained though the invention is not identically disclosed or described as set forth in section 102 of this title, if the differences between the subject matter sought to be patented and the prior art are such that the subject matter as a whole would have been obvious at the time the invention was made to a person having ordinary skill in the art to which said subject matter pertains. Patentability shall not be negatived by the manner in which the invention was made.

8. This application currently names joint inventors. In considering patentability of the claims under 35 U.S.C. 103(a), the examiner presumes that the subject matter of the various claims was commonly owned at the time any inventions covered therein were made absent any evidence to the contrary. Applicant is advised of the obligation under 37 CFR 1.56 to point out the inventor and invention dates of each claim that was not commonly owned at the time a later

invention was made in order for the examiner to consider the applicability of 35 U.S.C. 103(c) and potential 35 U.S.C. 102(e), (f) or (g) prior art under 35 U.S.C. 103(a).

9. Claims 1, 3, 7-9, 11, 12, 15-17 and 22 are rejected under 35 U.S.C. 103(a) as being unpatentable over Fujio et al., 2000 (Journal of Immunology, Vol. 165, p. 528-532) in view of Tsuji et al., April 2003 (Cancer Science, Vol. 94, No. 4, p. 389-393), Kessels et al., 2001 (Nature Immunology, Vol. 2, No. 10, p. 957-961) and Nishimura, T., 2000 (Cancer Treatment and Host, Vol. 12, No. 4, p. 363-373, IDS-CL).

Claims 1, 3, 7, 8 and 22 are directed to a process of preparing cells for cell therapy comprising inducing helper T cells that have a nonspecific antitumor activity, and imparting antigen specificity to the helper T cells by transducing the helper T cells with a T cell receptor gene that recognizes a cancer-associated antigen, wherein the TCR gene is a MHC class I-restricted T cell receptor gene. Claims 7 and 8 specify further purifying the Th1 cells to which antigen specificity has been imparted by using antibody-bearing magnetic beads. Claim 22 specifies the T cell receptor gene is isolated from a tumor specific human cytotoxic T cell clone. Claim 9, 11, 12 and 15-17 are directed to a process of preparing cells for cell therapy comprising inducing Th1 cells and Tc1 cells having a nonspecific antitumor activity and imparting antigen specifically to the Th1 cells and Tc1 cells by transducing the Th1 cells and Tc1 cells with a TCR gene that recognizes a cancer-associated antigen. Claims 11 and 12 specify transducing with MHC class I-restricted TCR gene and MHC class II-restricted TCR gene, respectively. Claims 15 and 16 specify further purifying the Th1 cells and Tc1 cells to which antigen specificity has been imparted by using antibody-bearing magnetic beads. Claim 17 specifies further comprising a step of mixing the separated Th1 cells and Tc1 cells in any given proportion.

Fujio teaches that "transfer of the alphabeta TCR genes into T lymphocytes will provide a means to enhance Ag-specific immunity by increasing the frequency of tumor- or pathogen-specific T lymphocytes. Fujio co-transfect TG40 cells, a TCR-negative mouse T cell line, with retroviral vector expressing either of the class II MHC-restricted alpha or beta TCR gene specific for chicken OVA and results in expression of the clonotypic TCR in 44% of the CD4+ T cells (helper T cells). "The transduced cells showed a remarkable response to OVA323-339 peptide in the in vitro culture system ... Adoptive transfer of the TCR-transduced cells in mice induced the Ag-specific delayed-type hypersensitivity in response to OVA323-339 challenge" (e.g. abstract, p. 528, right column, last paragraph). The TG40 cells are considered induced helper T cells. The chicken OVA is considered a cancer-associated antigen.

Fujio does not specifically teach transducing Th1 cells with MHC class I-restricted TCR gene, transducing both Th1 and Tc1 cells with TCR gene. Fujio also does not specifically teach separating the Th cells or Th1 and Tc1 cells with antibody-bearing magnetic beads or mixing separated Th1 cells and Tc1 cells in any proportion. Fujio does not specifically teach using TCR gene isolated from a tumor specific human cytotoxic T cell clone.

Tsuji discloses preparation of nonspecific Tc1 cells, naïve CD8+ T cells from C57BL/6 mouse spleen and activation of those cells by 2ug/ml plate bound anti-CD3 mAb under Tc1, Tc2 or neutral condition (e.g. p. 389, right column). Antigen-nonspecific CD8+ T cells were polyclonally expanded in the presence of IL-2, Th1 cytokines (IFN-gamma and IL-12) and anti-IL-4 mAb. The polyclonally activated CD8+ cells were transduced by retrovirus expressing 2C TCR alpha or 2C TCR beta chain to generate antigen-specific cytotoxic T lymphcytes (CTL).

The 2C-TCR gene-modified antigen specific Tc1 cells exhibit antitumor activity both in vitro and in vivo (e.g. abstract).

Kessels teaches that "the antigen specificity of T lymphocytes is dictated solely by the T cell receptor (TCR) alpha and beta chains. Consequently, genetic transfer of TCR chains may be an appealing strategy with which to impose a desirable virus- or tumor-antigen specificity onto cytotoxic or helper T cell populations" (e.g. abstract). Kessels introduces F5 TCR chains into mouse spenocytes by retroviral infection and 5-15% of the total CD8+ T cells (cytotoxic T cells) expressed the F5 TCR. The F5 TCR-transduced T cells show a pronounced NP(366-374)-specific effector function and the retroviral introduction of TCRs leads to rapid generation of antigen-specific T cell immunity (e.g. p. 958, left column, 2nd paragraph). Kessels also introduces F5 TCR chains into total spleen cells, including CD4+ T cells (helper T cells) with pMX-F5 virus and 5-10% of the CD4+ T cells expressed the F5 receptor. However, "in contrast to the marked expansion of F5 TCR CD8+ T cells upon influenza A/NT/60/68 infection, no expansion of F5 TCR CD4+ T cells was detected…This lack of expansion of TCR-transduced CD4+ T cells was consistent with the idea that CD4 coreceptor binding and signaling is required for proper T cell activation; it suggests that, for the simultaneous induction of CD4+ T cell immunity, coapplication of retrovirus encoding MHC class II-restricted TCRs may be considered" (e.g. p. 959, left column, 2nd paragraph).

Nishimura teaches that it is difficult to maximize activation of antitumor immunity in vivo only by MHC class I-associated peptide, activation of class II-restricted helper T (Th) cells is also required for induction of CTL which has recognized class I-associated tumor peptide (e.g. p. 363 (2-1 in the submitted copy filed 10-12-06).

It would have been prima facie obvious for one of ordinary skill in the art at the time of the invention to transduce Th1 cells with MHC class I-restricted TCR gene, or to transduce both Th1 and Tc1 cells with either MHC class I or class II-restricted TCR gene because Fujio teaches generation of MHC class II-restricted TCR transduced CD4+ T cells (helper T cells) that has antigen-specific immunity, Tsuji teaches transducing Tc1 cells with a retrovirus expressing 2C TCR gene, which is a MHC class I-restricted TCR, and the transduced Tc1 cells exhibit anti-tumor activity, and Kessels teaches retroviral transduction of both CD8+ T cells and CD4+ T cells with MHC class I-restricted TCR (F5 TCR) and that CD4+ T cells (helper T cells) may require expression of both MHC class I and class II-restricted TCR for the induction of CD4+ T cell immunity against tumor antigen, and Nishimura shows that activation of class II-restricted helper T cells is required for induction of CTL (cytotoxic T cells) which recognized class I-restricted tumor peptide. One of ordinary skill would be motivated to transduce Th1 and Tc1 cells with either MHC class I or class II-restricted TCR gene in order to optimize the tumor antigen specificity of the Th1 cells or Tc1 cells. It would have been obvious for one of ordinary skill in the art to separate the Th cells or Th1 and Tc1 cells with antibody-bearing magnetic beads or mixing separated Th1 cells and Tc1 cells in any proportion because it was known in the art to use antibody-bearing beads to separate cells with different antigen on the cell surface and determining various mixing proportion of Th1 cells and Tc1 cells would be routine optimization of a result effective variable. It would have been prima facie obvious to one of ordinary skill in the art to use TCR gene isolated from tumor specific human cytotoxic T cell clone because Fujio teaches using cDNA for TCR alpha and beta chains isolated from a cDNA library of DO11.10 TCR Tg splenocytes and since they are all TCR genes one would try to isolate the TCR genes

from various sources so as to transduce Th1 or Tc1 cells with retroviral vector to provide a desirable virus- or tumor-antigen specificity onto the Th1 and Tc1 cells.

One having ordinary skill in the art at the time the invention was made would have been motivated to do so in order to generate 2C-TCR gene-modified antigen specific Tc1 cells for exhibiting antitumor activity as taught by Tsuji or to impose a desirable virus- or tumor-antigen specificity onto cytotoxic or helper T cell populations as taught by Kessels with reasonable expectation of success.

10. Claims 1 and 5 are rejected under 35 U.S.C. 103(a) as being unpatentable over Fujio et al., 2000 (Journal of Immunology, Vol. 165, p. 528-532) in view of Gaiger et al., 2008 (US Patent No. 7323181 B2).

Claims 1 and 5 are directed to a process of preparing cells for cell therapy comprising inducing helper T cells that have a nonspecific antitumor activity, and imparting antigen specificity to the helper T cells by transducing the helper T cells with a T cell receptor gene that recognizes a cancer-associated antigen. Claim 5 specifies the cancer-associated antigen is WT1.

Fujio teaches that "transfer of the alphabeta TCR genes into T lymphocytes will provide a means to enhance Ag-specific immunity by increasing the frequency of tumor- or pathogen-specific T lymphocytes. Fujio co-transfect TG40 cells, a TCR-negative mouse T cell line, with retroviral vector expressing either of the class II MHC-restricted alpha or beta TCR gene specific for chicken OVA and results in expression of the clonotypic TCR in 44% of the CD4+ T cells (T helper cells). "The transduced cells showed a remarkable response to OVA323-339 peptide in the in vitro culture system ... Adoptive transfer of the TCR-transduced cells in mice induced the

Ag-specific delayed-type hypersensitivity in response to OVA323-339 challenge" (e.g. abstract, p. 528, right column, last paragraph). The TG40 cells are considered induced helper T cells. The chicken OVA is considered a cancer-associated antigen.

Fujio does not specifically teach using cancer-associated antigen Wilms' tumor 1 (WT1).

Gaiger teaches that "T cells specific for WT1 can kill cells that express WT1 protein. Introduction of genes encoding T-cell receptor (TCR) chains for WT1 are used as a means to quantitatively and qualitatively improve response to WT1 bearing leukemia and cancer cells" (e.g. column 26, last paragraph). Non-specific T cells can be transfected with a polynucleotide encoding TCRs specific for a polypeptide described herein to render the host cell specific for the polypeptide. The hose cells can be used for adoptive immunotherapy of WT associated cancer (e.g. column 28, 1st paragraph).

It would have been prima facie obvious for one of ordinary skill in the art at the time of the invention to transduce Th1 cells with a TCR gene for WT1 because Gaiger teaches transducing T cells with a gene encoding TCR chains for WT1 to render the host cell specific for adoptive immunotherapy of WT associated cancer.

One having ordinary skill in the art at the time the invention was made would have been motivated to do so in order to produce T cells expressing TCR specific for WT1 polypeptide for adoptive immunotherapy of WT associated cancer as taught by Gaiger with reasonable expectation of success.

11. Claims 9 and 13 are rejected under 35 U.S.C. 103(a) as being unpatentable over Kessels et al., 2001 (Nature Immunology, Vol. 2, No. 10, p. 957-961) in view of Gaiger et al., 2008 (US Patent No. 7323181 B2).

Claims 9 and 13 are directed to a process of preparing cells for cell therapy comprising inducing Th1 cells and Tc1 cells having a nonspecific antitumor activity and imparting antigen specifically to the Th1 cells and Tc1 cells by transducing the Th1 cells and Tc1 cells with a TCR gene that recognizes a cancer-associated antigen. Claim 13 specifies the cancer-associated antigen is WT1.

Kessels teaches that "the antigen specificity of T lymphocytes is dictated solely by the T cell receptor (TCR) alpha and beta chains. Consequently, genetic transfer of TCR chains may be an appealing strategy with which to impose a desirable virus- or tumor-antigen specificity onto cytotoxic or helper T cell populations" (e.g. abstract). Kessels introduces F5 TCR chains into mouse spenocytes by retroviral infection and 5-15% of the total CD8+ T cells (cytotoxic T cells) expressed the F5 TCR. The F5 TCR-transduced T cells show a pronounced NP(366-374)-specific effector function and the retroviral introduction of TCRs leads to rapid generation of antigen-specific T cell immunity (e.g. p. 958, left column, 2nd paragraph). Kessels also introduces F5 TCR chains into total spleen cells, including CD4+ T cells with pMX-F5 virus and 5-10% of the CD4+ T cells (helper T cells) expressed the F5 receptor. However, "in contrast to the marked expansion of F5 TCR CD8+ T cells upon influenza A/NT/60/68 infection, no expansion of F5 TCR CD4+ T cells was detected…This lack of expansion of TCR-transduced CD4+ T cells was consistent with the idea that CD4 coreceptor binding and signaling is required for proper T cell activation; it suggests that, for the simultaneous induction of CD4+ T cell

immunity, coapplication of retrovirus encoding MHC class II-restricted TCRs may be considered" (e.g. p. 959, left column, 2nd paragraph). Imparting antigen specificity to the Th1 cells and Tc1 cells only requires transducing the Th1 cells and Tc1 cells with a TCR gene that recognizes a cancer-associated antigen. F5 TCR is a MHC class I-restricted TCR.

Kessels does not specifically teach using cancer-associated antigen Wilms' tumor 1 (WT1).

Gaiger teaches that "T cells specific for WT1 can kill cells that express WT1 protein. Introduction of genes encoding T-cell receptor (TCR) chains for WT1 are used as a means to quantitatively and qualitatively improve response to WT1 bearing leukemia and cancer cells" (e.g. column 26, last paragraph). Non-specific T cells can be transfected with a polynucleotide encoding TCRs specific for a polypeptide described herein to render the host cell specific for the polypeptide. The hose cells can be used for adoptive immunotherapy of WT associated cancer (e.g. column 28, 1st paragraph).

It would have been prima facie obvious for one of ordinary skill in the art at the time of the invention to transduce Th1 cells with a TCR gene for WT1 because Gaiger teaches transducing T cells with a gene encoding TCR chains for WT1 to render the host cell specific for adoptive immunotherapy of WT associated cancer.

One having ordinary skill in the art at the time the invention was made would have been motivated to do so in order to produce T cells expressing TCR specific for WT1 polypeptide for adoptive immunotherapy of WT associated cancer as taught by Gaiger with reasonable expectation of success.

Applicant argues that one of ordinary skill in the art could not have reasonably predicted that the same MHC class restricted TCR gene could have been used to obtain both helper cells and cytotoxic T1 cells because these cells have different signaling requirements. TCR of CTL recognizes an antigenic substance presented on a MHC class I molecules of a target cell and TCR of a helper T cell res an antigenic substance presented on a MHC class II molecule of monocytes/macrophage (amendment, p. 8-10). This is not found persuasive because of the reasons set forth above under 35 U.S.C. 102(b) and 103(a) rejections. Kessels teaches retroviral transduction of both CD8+ T cells (cytotoxic T cells) and CD4+ T cells (helper T cells) with MHC class I-restricted TCR (F5 TCR), therefore, it would be obvious to one of ordinary skill in the art to use the same MHC class restricted TCR gene to obtain both helper cells and cytotoxic T1 cells. Further, the only requirement to impart antigen specificity to the T cells is to introduce a TCR gene into the T cells and it is prima facie obvious to one of ordinary skill in the art in view of the teachings of Fujio, Tsuji, Keesles and Nishmura.

Conclusion

No claim is allowed.

Any inquiry concerning this communication or earlier communications from the examiner should be directed to Shin-Lin Chen whose telephone number is (571) 272-0726. The examiner can normally be reached on Monday to Friday from 9:30 am to 6 pm.

If attempts to reach the examiner by telephone are unsuccessful, the examiner's supervisor, Peter Paras can be reached on (571) 272-4517. The fax phone number for this group is (571) 273-8300.

Any inquiry of a general nature or relating to the status of this application or proceeding should be directed to (571) 272-0547.

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Shin-Lin Chen, Ph.D.
/Shin-Lin Chen/
Primary Examiner
Art Unit 1632